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DR. JAMES G. CARR, Chicago, *Editor*

DR. GILBERT H. MARQUARDT, Chicago, *Associate Editor*

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THE ELECTROCARDIOGRAM IN PERICARDITIS

The discovery of the electrocardiographic changes which occur with and which at times are diagnostic of pericarditis is a comparatively recent one. The first article on this subject appeared in the literature in 1923 and it is only since 1929 that progress has been made, through experimental and clinical studies, in the definition of the various features of the electrocardiogram in this condition and in the suggestion of possible etiologic mechanisms for them. Schwab and Herrmann presented an excellent review of the literature; this will be brought up to date in an article by the authors, which will appear in the near future.

The recognition of the electrocardiographic changes associated with pericarditis has a twofold importance: first, as an aid in the diagnosis of uncomplicated acute and chronic pericarditis and in distinguishing these forms of pericarditis from other types of cardiac disease, particularly cardiac infarction; second, as an aid to the correct evaluation of the electrocardiogram when pericarditis complicates such conditions as cardiac infarction, acute rheumatic fever, operations on the heart, experimental coronary artery occlusion, uremia, and possibly pneumonia. It has often been said that the diagnosis of acute pericarditis is more often missed than made. Purulent pericarditis is a condition which calls for early surgical intervention. Acute pericarditis occurring after various operations may be confused easily with cardiac infarction. Chronic constrictive pericarditis now is so successfully treated by pericardiectomy that its detection and the differential diagnosis of this condition and hepatic cirrhosis, mitral stenosis, and other conditions are of great importance. In experimental coronary occlusion, unless the electrocardiographic effect of the pericarditis is recognized, there will result confusion as to the significance of the findings, particularly regarding their localizing value. Likewise, in clinical cases of cardiac infarction a complicating pericarditis may produce elevation of the RS-T segment in all the standard leads and later inversion of the T wave in these leads, which will mask the characteristic T_1 and T_4 pattern. In acute rheumatic fever certain findings in the electrocardiogram which previously were thought to indicate rheumatic myocarditis in the usual sense or which even were ascribed to rheumatic involvement of the coronary arteries, are now recognized as having their origin in a complicating pericarditis. In the terminal stage of uremia the ominous signs of pericarditis may appear first in the electrocardiogram. While minor electrocardiographic changes may occur in cases of pneumonia, the presence of well-marked elevation of the RS-T segment should lead to the suspicion of a complicating pericarditis.

Summary of the Electrocardiogram in Cases of Acute and Subacute Pericarditis

Changes in the RS-T segment.—In tracings of normal persons the RS-T segment arises on or a few tenths of a millimeter above or below the iso-electric line. Occasionally, it may be elevated 0.5 mm. and in rare instances elevations of 1.3 mm. have been found. The degree of variation from the usual position is somewhat dependent upon the height of the QRS complex so that it has been said that when the latter is comparatively low an elevation of more than 0.5 mm. may be considered abnormal. In acute pericarditis an abnormal elevation of the RS-T segment may occur in all the standard leads, in the combined leads I and II, or only in lead I. Frequently, leads I and II are elevated while lead III is iso-electric or insignificantly elevated. The reciprocal depression of the RS-T segment in lead III, which usually is seen in association with elevation of the RT segment in lead I in the T_1 pattern of anterior infarction does not occur in pericarditis. The greatest elevation of the RS-T segment often occurs in lead II, in contrast with the usual findings in anterior or posterior infarction. There may be, although it is frequently absent, a depression of the RS-T segment of more than the normal amount of 2.0 mm. in precordial leads taken by the Wolferth method.

The contour of this segment, while it is elevated, is somewhat different from that encountered in cardiac infarction. Since in the early stages of pericarditis the T wave is upright and sometimes exaggerated, the R-T segment in this stage is concave upward, as contrasted with the contour which is convex upward in infarction. In other instances it rises in a straight line as an inclined plane from its origin to end in an upright T which may be exaggerated. The elevation frequently disappears after only a few days, and thus usually is of shorter duration than the elevation in infarction.

Changes in the T waves.—Early in acute pericarditis the T waves are upright and sometimes exaggerated. They may then change to any position between this and the deeply inverted form, which previously was thought to occur only in cardiac infarction. In a good share of cases the T waves remain upright although a decrease in voltage usually appears. In some cases the T wave may be inverted in all of the conventional leads, a situation rarely seen in other types of cardiac disease. The inversion appears most commonly in the first two leads and consequently the opposite direction of the T waves in leads I and III seen in the late T_1 type of electrocardiogram may be observed although the T wave in the third lead is not so sharply peaked and exaggerated as it may be at a certain stage after infarction of the anterior apical region of the heart. A reversal in direction of T wave in the precordial lead may occur, and, in general, when significant changes in the T wave are seen in the standard leads, T_4 will show some modification in voltage or direction. Frequently, when the T wave is of low voltage in the standard leads it may assume a characteristic dome contour with a gradual slight rise, flattened summit, and gradual fall. It reverts to its normal direction and contour after varying lengths of time, usually considerably sooner than in cardiac infarction.

Changes in the voltage of QRS.—In many cases of pericarditis with effusion there is a decrease in the voltage of the QRS complexes in the standard leads, often of a degree falling within the classification of low voltage. The precordial lead may or may not be similarly affected. However, considerable amounts of fluid may be present without the appearance of low voltage, and removal of fluid from the pericardial sac does not consistently increase the voltage. Although the voltage is low there is seldom the marked slurring or notching of the QRS complexes that are seen in many cases in which low voltage is due to marked myocardial injury.

Q wave in pericarditis.—Although small Q waves may be present in the standard leads, the typical Q_1 or Q_3 patterns of cardiac infarction are not seen. The Q wave in the precordial lead taken by the Wolferth method is present, except in rare instances.

Summary of the Electrocardiogram of Chronic Pericarditis

The following types of chronic pericarditis are known to produce electrocardiographic changes which suggest pericardial (subepicardial myocardial) involvement: chronic constrictive pericarditis with or without calcification, chronic tuberculous pericarditis, and carcinomatous involvement of the pericardium. It seems probable that the type of chronic pericarditis which is frequently seen as an incidental finding at necropsy produces no recognizable change in the electrocardiogram.

Low voltage of the QRS complex in all leads is present in the great majority of cases. The P and T waves are also often of low voltage. The T wave may be iso-electric or inverted in one, two or three leads. In about 50 per cent of cases of constrictive pericarditis the T wave in precordial leads taken by the Wolferth method is upright. The QRS complexes may be slightly slurred but they do not exhibit the marked slurring or notching seen in low voltage associated with advanced myocardial degeneration. Auricular fibrillation occurs rather frequently. In chronic tuberculous pericarditis the QRS voltage and T waves vary with the course of the underlying pathologic process.

Pericarditis Complicating Coronary Occlusion

This may result in elevation of the RS-T segment in the three standard leads and later in inversion of the T waves in these leads, thus obscuring the characteristic patterns of anterior and posterior infarction. If tracings are obtained within the first day or two after the infarction has occurred the effect of the pericarditis may not yet be present since it usually develops after three or four days. Also, after varying lengths of time the changes due to pericarditis may disappear and leave a typical late T-wave pattern by which the situation of the previous infarction may be determined.

Pericarditis Complicating Operations Upon the Heart

It has been assumed generally that the electrocardiographic changes which occurred after operation for wounds of the heart were due entirely to the myocardial injury produced by the wound, which sometimes necessitated suture of the coronary arteries. Inspection of the electrocardiogram made in these cases, particularly in cases in which there was no involvement of the coronary arteries, reveals many of the changes characteristic of pericarditis. Operations to increase the blood supply of the heart, such as cardio-omentopexy or the injection of irritant solutions into the pericardial sac, produce a pericarditis that results in the usual alteration of the electrocardiogram.

Pericarditis Complicating Uremia

If a pericardial effusion occurs, low voltage of the QRS complexes may be present. When the preceding electrocardiograms show a marked left ventricular strain with depression of the RS-T interval in lead I and inverted T wave in leads I, or I and II, the pericarditis may affect the tracing by lessening or abolishing the RS-T depression that at times may lead to well-marked elevation of this segment in one or more of the conventional leads.

Theories Concerning the Etiology of the Electrocardiographic Changes in Pericarditis

It is generally believed that the low voltage of the QRS complexes in acute pericarditis is due to the short-circuiting effect of the pericardial effusion upon the action currents developed by the excitatory and contractile processes. The low voltage of the QRS complexes in cases of chronic constrictive pericarditis has never been explained. The lack of correspondence between the amount of effusion and the voltage of the QRS complex as well as the varying effect of pericardial paracentesis upon its voltage suggests that some factor other than the short-circuiting effect may be present.

The elevations of the RS-T segment may be produced experimentally by introducing fluid into the pericardial sac. They correspond to the resulting decrease in aortic and carotid blood pressures. This suggests that an impairment of circulation in the coronary arteries with generalized myocardial ischemia may be the cause of these elevations. Other experiments have shown that unless the pressure in the pericardial sac is maintained by increasing the volume of the injected fluid the pressure will soon decrease as a result of stretching of the pericardium, and that the electrocardiogram will return to normal. It is probable, therefore, that this mechanism obtains clinically only when the effusion accumulates so rapidly or is of such a volume that the limit of relaxation of the pericardium is reached. Hemopericardium and purulent pericarditis are conditions in which this may reasonably occur. It is a common clinical experience that elevations of the RS-T segment, of considerable degree, may appear early in the course of pericarditis when there are no signs of effusion, and that the elevations may disappear during the period when fluid is known to be accumulating. Also, the removal of fluid has no consistent effect upon the elevation. There would seem to be another factor present in many cases. Experimental studies and a recent report of a series of cases studied clinically and pathologically have shown that the appearance of electrocardiographic changes seems to depend upon the penetration of the inflammatory process through the visceral pericardium into the subepicardial myocardium. It has been demonstrated previously that under experimental conditions the T wave returns to normal simultaneously with the complete replacement of inflammatory exudate in the subepicardial myocardium by fibrous tissue. Subepicardial myocarditis is probably the important factor responsible for the alteration of the electrocardiogram in the great majority of cases.

Incidence of Electrocardiographic Changes in Pericarditis

The incidence of electrocardiographic changes in cases of pericarditis has been reported to vary from 35 per cent to more than 80 per cent. In a preliminary review of fifty-three cases of all types of pericarditis which we have observed at The Mayo Clinic, there were five cases in which the electrocardiograms were regarded as pathognomonic of pericarditis; in twenty-two cases the electrocardiograms were diagnostic or strongly suggestive of pericarditis when the clinical circumstances were known; in ten cases there were changes which would lead only to the suspicion that pericarditis might be present; in sixteen cases there were no changes which were suggestive of pericarditis. There is no doubt that serial tracings in all cases would have greatly increased the incidence of diagnostic findings.

Paul H. Noth, M.D.
and
Arlie R. Barnes, M.D.
Rochester, Minnesota